Heat-Processed Scutellariae Radix Protects Hepatic Inflammation through the Amelioration of Oxidative Stress in Lipopolysaccharide-Induced Mice.

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Abstract

The present study evaluated the effects of heat-processed Scutellariae Radix (Scutellaria baicalensis) on lipopolysaccharide (LPS)-induced liver injury in mice. Scutellariae Radix heat-processed at 160[Formula: see text]C or 180[Formula: see text]C was orally administered at a dose of 100 mg/kg body weight for three days before the intraperitoneal injection of LPS, and the effects were compared with those of vehicle-treated LPS administered to control mice. The administration of Scutellariae Radix decreased the elevated serum monocyte chemotactic protein-1 (MCP-1), interleukin-6 (IL-6), reactive oxygen species (ROS), nitrite/nitrate, peroxynitrite, and hepatic functional parameters, and reduced the increased ROS in the liver. The augmented expressions of hepatic oxidative stress and inflammation-related proteins, phospho-p38, phosphorylated extracellular signal-regulated kinase, phosphorylated c-Jun N-terminal kinase, nuclear factor-[Formula: see text] B p65, activator protein-1, cyclooxygenase-2, inducible nitric oxide synthase, MCP-1, intercellular adhesion molecule-1, tumor necrosis factor-[Formula: see text], and IL-6, were downregulated by the heat-processed Scutellariae Radix. Hematoxylin-eosin staining showed that the increased hepatocellular damage in the liver of LPS-treated mice improved with the administration of heat-processed Scutellariae Radix. Overall, the ameliorative effects of Scutellariae Radix were superior to those when heat-processed at 180[Formula: see text]C. Our results indicate that heat-processed Scutellariae Radix acts as an anti-inflammatory agent by ameliorating oxidative stress in the liver of mice with LPS-induced liver injury.